AMENDMENTS TO THE CLAIMS

Please amend the claims as follows:

This listing of claims will replace all prior versions, and listing, of claims in the application:

Listing of Claims:

Claim 1 (currently amended) A method for treatment of heart failure comprising attenuating PLB-induced cardiac SR Ca^{2+} ATPase (SERCA2a) inhibition and enhancing contractility in a heart comprising ~~inducing phospholamban deficiency, wherein~~

(a) providing a compound comprising an exogenous dominant negative phospholamban (PLB) protein functionally attached to a transport ~~penetratin~~ peptide; and

(b) contacting the heart with the compound, thereby attenuating PLB-induced cardiac SR Ca^{2+} ATPase (SERCA2a) inhibition and enhancing contractility in a heart to treat the heart failure ~~delivered to cardiac tissue induces phospholamban deficiency~~.

Claims 2 and 3 (canceled)

Claim 4 (currently amended) The method ~~for treatment of heart failure~~ of claim 19, wherein the mutations of PLB comprise sense ~~point~~ mutations.

Claims 5 to 11 (canceled)

Claim 12 (currently amended) A method for treatment of heart failure comprising enhancement of cardiac contractility by inhibition of phospholamban (PLB)-

[[PLB-]] sarcoplasmic reticulum calcium ATPase (SERCA2a) interaction comprising

(a) providing ~~wherein~~ an exogenous dominant negative PLB protein functionally attached to a transport ~~penetratin~~ peptide; and

(b) delivering an effective amount of the compound ~~delivered~~ to cardiac tissue ~~is used~~ to inhibit interaction between PLB and SERC2a, thereby enhancing cardiac contractility and treating heart failure.

Claims 13 to 15 (canceled)

Claim 16 (currently amended) The method of claim 22, wherein the mutations of PLB comprise sense ~~point~~ mutations of PLB.

Claims 17 and 18 (canceled)

Claim 19 (currently amended) The method ~~for treatment of heart failure~~ of claim 1, wherein the exogenous dominant negative PLB protein comprises a PLB protein with mutations.

Claim 20 (currently amended) The method ~~for treatment of heart failure~~ of claim 19, wherein the exogenous dominant negative PLB protein comprises a truncated PBL protein.

Claim 21 (canceled)

Claim 22 (currently amended) The method ~~for treatment of heart failure~~ of claim 12, wherein the exogenous dominant negative PLB protein comprises a PLB protein with mutations.

Claim 23 (currently amended) The method ~~for treatment of heart failure~~ of claim 12, wherein the exogenous dominant negative PLB protein comprises a truncated PBL protein.

Claim 24 (new) A method for attenuating phospholamban (PLB)-induced cardiac SR Ca^{2+} ATPase (SERCA2a) inhibition in a heart cell or a muscle cell, comprising

(a) providing a compound comprising an expression construct comprising a coding sequence for a dominant negative PLB functionally linked to a promoter active in the heart or the

muscle cell, and the dominant negative PLB binds to wild-type PLB; and

(b) contacting the heart cell or the muscle cell with an effective amount of the compound, thereby attenuating phospholamban (PLB)-induced cardiac SR Ca^{2+} ATPase (SERCA2a) inhibition in the heart cell or the muscle cell.

Claim 25 (new) A method for increasing cardiac SR Ca^{2+} ATPase (SERCA2a) activity in a heart cell or a muscle cell,

(a) providing a compound comprising an expression construct comprising a coding sequence for a dominant negative phospholamban (PLB) functionally linked to a promoter active in the heart or the muscle cell, and the dominant negative PLB binds to wild-type PLB in the heart or the muscle cell; and

(b) contacting the heart cell or the muscle cell with an effective amount of the compound, thereby attenuating PLB-induced SERCA2a inhibition and increasing SERCA2a activity in the cell.

Claim 26 (new) The method of claim 24 or claim 25, wherein the heart cell is a cardiac myocyte.

Claim 27 (new) The method of claim 24 or claim 25, wherein the muscle cell is a smooth muscle cell.

Claim 28 (new) The method of claim 24 or claim 25, wherein the contacting is *in vitro*.

Claim 29 (new) The method of claim 24 or 25, wherein the contacting is *in vivo*.

Claim 30 (new) The method of claim 24 or 25, wherein the expression construct comprising a coding sequence for a dominant negative PLB further comprises a coding sequence for a penetratin peptide.

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Claim 31 (new) A method for enhancing contractility in a heart comprising

(a) providing a compound comprising an expression construct comprising an expression construct comprising a coding sequence for a dominant negative phospholamban (PLB) functionally linked to a promoter active in the heart, and the dominant negative PLB binds to wild-type PLB; and

(b) contacting the heart with an effective amount of the compound, thereby attenuating PLB-induced cardiac SR Ca^{2+} ATPase (SERCA2a) inhibition and enhancing contractility in the heart.

Claim 32 (new) A method for increasing heart activity comprising

(a) providing a compound comprising an expression construct comprising a coding sequence for a dominant negative phospholamban (PLB) functionally linked to a promoter active in the heart, and the dominant negative PLB binds to wild-type PLB; and

(b) contacting the heart with an effective amount of the compound, thereby attenuating PLB-induced cardiac SR Ca^{2+} ATPase (SERCA2a) inhibition and increasing heart activity.

Claim 33 (new) A method for treating heart failure comprising

(a) providing a compound comprising an expression construct comprising a coding sequence for a dominant negative phospholamban (PLB) functionally linked to a promoter active in the heart, and the dominant negative PLB binds to wild-type PLB; and

(b) contacting the heart with an effective amount of the compound, thereby attenuating PLB-induced cardiac SR Ca^{2+} ATPase (SERCA2a) inhibition, enhancing contractility or relaxation in the heart and treating the heart failure.

Claim 34 (new) The method of claim 31, claim 32 or claim 33, wherein the expression construct comprises an adenoviral expression construct.

Claim 35 (new) The method of claim 31, claim 32 or claim 33, wherein the dominant negative phospholamban (PLB) has an altered amino acid sequence as compared to wild-type PLB.

Claim 36 (new) The method of claim 31, claim 32 or claim 33, wherein the dominant negative phospholamban (PLB) is a truncated PLB.

Claim 37 (new) The method of claim 36, wherein the truncated PLB comprises SEQ ID NO:8.

Claim 38 (new) The method of claim 36, wherein the truncated PLB comprises a PLB cytoplasmic domain.

Claim 39 (new) The method of claim 31, claim 32 or claim 33, wherein the dominant negative phospholamban (PLB) has an altered amino acid sequence as compared to wild-type PLB.

Claim 40 (new) The method of claim 1 or claim 12, wherein the exogenous dominant negative PLB protein is linked to the transport peptide by a covalent linkage.

Claim 41 (new) The method of claim 40, wherein the covalent linkage comprises a polylysine, a single peptide bond or a disulfide bond.

Claim 42 (new) The method of claim 41, wherein the polylysine comprises a branched polylysine.

Claim 43 (new) The method of claim 40, wherein the transport peptide comprises an antennapedia transport peptide or a penetratin.

Claim 44 (new) The method of claim 40, wherein the antennapedia transport peptide comprises SEQ ID NO:7.

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Claim 45 (new) The method of claim 1 or claim 12, wherein the exogenous dominant negative PLB protein comprises the first 16 residues of SEQ ID NO:8; SEQ ID NO:17; SEQ ID NO:18; or SEQ ID NO:19.

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